UMNL vs LMNL

INTENDED LEARNING OBJECTIVES (ILOs)

By the end of this lecture the student will be able to:

1. Describe upper and lower motor neuron
2. Compare upper and lower motor neuron lesions
3. Explain babinski's sign
4. Explain clonus
5. Explain lengthening reaction (clasp knife)

UMNs:

- Are neurons in cerebral cortex and brain stem that activate lower motor neurons. Their axons form the descending motor pathways.

- For movement of head muscles are neurons of CMAs and their axons CBT only.
- For movement of Body muscles are neurons of CMAs and their axons CST + COEP
- CST only --- on → alpha..... Fine discrete movement.
- COEP----- on → alpha rubro-sp..... .Gross Movement. → gamma reticule-sp..... Gross Movement.

LMNs:

- Are spinal and cranial motor neurons that directly innervate skeletal muscles. Their axons proceed through peripheral somatic nerves to innervate skeletal muscles.

- For movement of head muscles are nuclei and axons of cranial nerves.
- For movement of Body muscles are alpha motor neurons.

So UMNL or LMNL causes loss of both fine and gross movements, i.e. Paralysis.

UMNL

Definition: It is damage of descending motor tracts (From c.c. to A.H.C.) both Pyramidal + COEP.

Sites:

1. CMAs not common, causes contra lateral Monoplegia (Extensive representation).

2. In post, limb of internal capsule.
3. Brain Stem causes Contra lateral Hemiplegia.
4. In spinal cord the only site in which the effects are on the same side.

In post, limb of internal capsule
Cause: vascular, hemorrhage or thrombosis of Lenticulo-striate artery.
Acute UMNL: Immediate Effects (Cerebral Stroke)

1. Contra-lateral flaccid paralysis for 2-6 weeks affecting opposite UL + LL + Lower facial Ms.+ 1/2 of tongue.
2. Loss of superficial reflexes.
3. Positive Babinski's sign.
4. MSR: Hypotonia (Flaccidity) and areflexia due to loss of excitatory effect of pyramidal fibers on alpha motor neurons.
5. Contra-lateral Hemi anesthesia.

Chronic stage: (spastic paralysis) Permanent UMNL:
A) Motor Disturbances:
1. MSR increased activity due to reversed supra-spinal balance from inhibitory to Excitatory; i.e. decreased activity of inhibitory reticular area and increased activity of excitatory reticular area, which causes:

a) Increased M.t.= spasticity. mainly in antigravity muscles → Flexors of UL → Extensors of LL
b) Hyperreflexia. Exaggerated tendon jerks.
c) Clonus (e.g. Ankle).
It is contraction-relaxation of calf muscles due to:
→ repeated m. spindle discharge.
→ stretch-inverse stretch Reflex sequence GTO.
d) Lengthening reaction (clasp knife effect).
Due to increased tension on tendon → sudden muscle relaxation. (Diff. Spasticity & Rigidity).

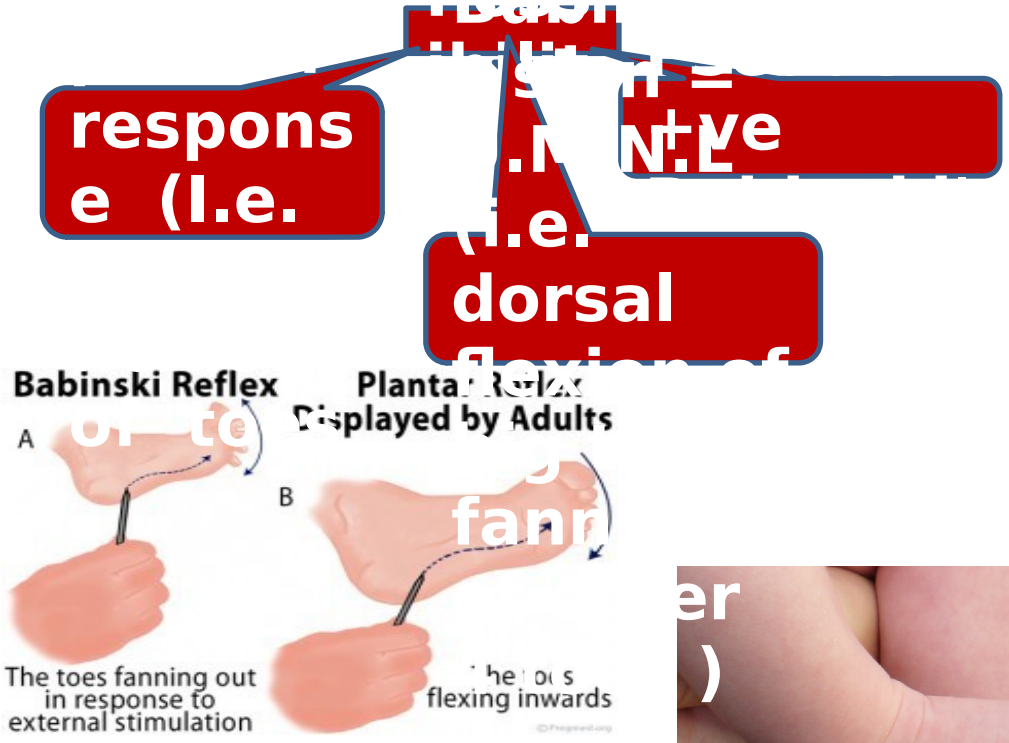
2. Slight recovery of gross movement, patient may walk, this is due to:

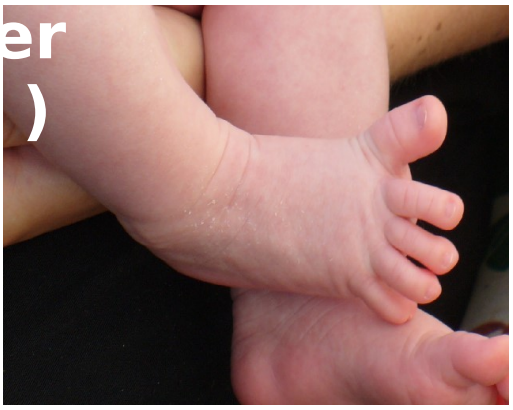
a) Ipsiateral CST.
b) Extra pyramidal tract from red n. (lower than int. capsule).
But still there is loss of muscle power, in voluntary acts.

3. Normal Muscle Bulk.
No immediate atrophy, as muscles are still innervated, contract reflexly.
4. Babinski's sign still positive.

N.B: Plantar response

- Center :- L5 – S1
- Afferent : Dorsal Root.
- Efferent :- Ventral Root





Pseudo +ve Babinski's sign: occur in:

a) Deep sleep.
b) Anesthesia .
c) Children (new Born).
d)

B) Sensory Disturbances:

1. Recovery of crude sensations: but fine sensations never recover.
2. Homonymous Hemianopia.
3. Decreased Auditory Acuity: But not complete deafness. WHY?

LMNL

Definition: It is lesion in A.H.C. or its axon.

Sites:

1. Spinal cord (damage of A.H.C.)

e.g. Syringomyelia or Poliomyelitis (one limb).

2. Damage of ventral Root

e.g. Traumatic spinal cord lesion.
3. Damage of peripheral nerves.
e.g. alpha compression of median nerve at wrist or Polyneuritis.
4. Damage of Ms. itself as in Myasthenia Gravis.

	Chronic UMNL	LMNL
1. Definition:	-Widespread	-Localized
	Contralateral	- Ipsi-lateral
	- Paralysis of movement	- Paralysis of separate Ms.
	- due to lesion in tract	- lesion in motor neuron.
2. Character	-Spastic paralysis	Flaccid Paralysis
3. Recovery	Never (no neurilemma)	May recover
4. Muscle state	Disuse Atrophy on long term	Immediate atrophy Muscle → fibrous tissue
5. Muscle Tone	Increased	Decreased (alpha damge)
6. Muscle power	Lost	lost
7. Sup. Reflexes	All Lost except +ve Babinski	All are lost
8. Deep Reflexes	Hypereflexia	Hyporeflexia
9. Reaction of Denervation	Absent	Present
10. Clonus	Present	Absent

N.B. Acute UMNL is exactly the same as LMNL except in: Widespread, +ve Babinski, No Atrophy.

Reaction of Denervation:
Occurs at least 10 days of lesion, in the period between loss of excitability of nerve and loss of excitability of muscle. It consists of:

1. Fasciculation: Rhythmic regular contractions affecting group of muscles, visible and palpable due to spontaneous discharge from nerve roots.

2. Fibrillation: Rhythmic contractions of separate muscle fibers not visible and not palpable, increase by warmth, decrease by cold, recorded by EMG due to increased sensitivity to circulating Ac.ch.

3. Abnormal response to electrical stimulation:

a) Normal response:

- Response to Faradic current > to galvanic
- CCC > ACC
- Cathodal closing cont. > Anodal closing cont.

b) Incomplete Reaction of Degeneration:

- ACC > CCC
-The muscle responds only to galvanic.

c) Complete R.D.

- Muscle completely degenerated.
- No response at all.

4. Loss of H. (Hoffman) reflex.

SUGGESTED TEXTBOOKS

Ganong's Review of Medical Physiology, twenty-fifth edition 2016, McGraw-Hill Education, chapter 12, from page 227 to 254